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Note: Further Evidence of an Inverse Relation between Mammalian Body Mass and Sensitivity to Radio-Frequency Electromagnetic Radiation

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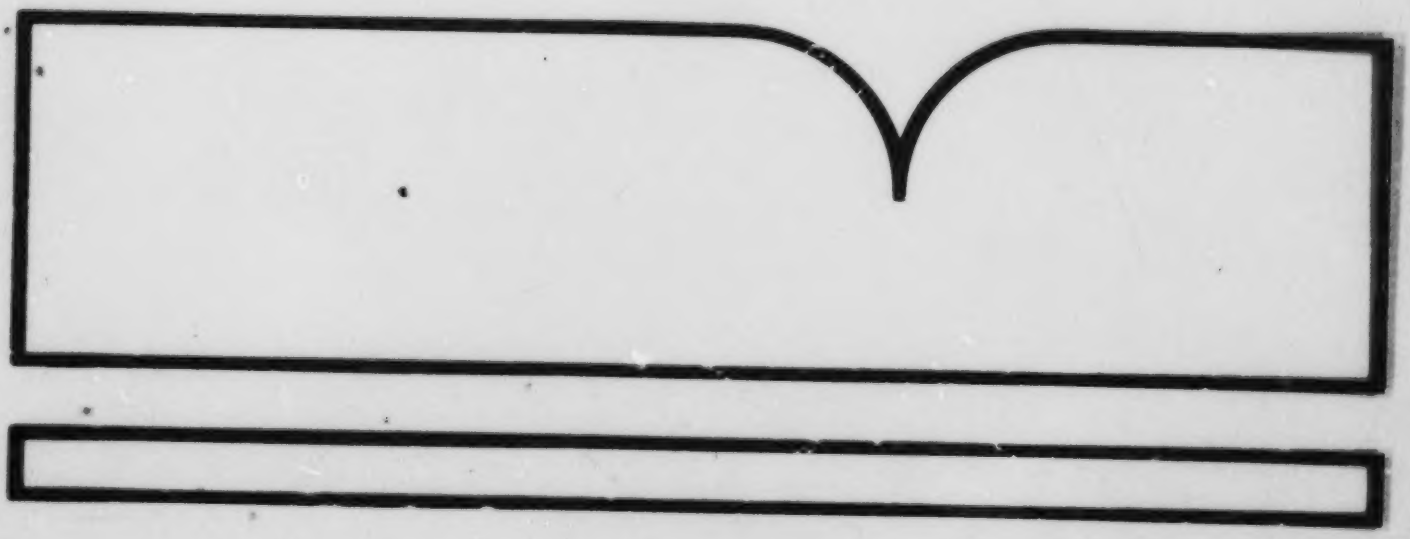

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16. ABSTRACT

The principal objectives of the paper under criticism were to interrelate the effects of ambient temperature and intensity of exposure to radiofrequency electromagnetic radiation (RFR) on the activity of asingle thermoregulatory effector, evaporative heat loss (EHL) and (ii) to ascertain if there is a relation between an animal's body mass and its sensitivity to thermalizing levesl of RFR. In the following response, I shall first explain the rationale and significance of interrelating the effects of T_a versus RFR; second, I shall attempt to demonstrate theoretically why there should be an inverse relation between body mass and sensitivity of the organism to RFR; and third, I shall report additional data that further demonstrate the priniciple of an inverse relation between body mass and SAR thresholds of physiological activity.

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JOURNAL ARTICLE

Note: Further Evidence of an Inverse Relation Between Mammalian Body Mass and Sensitivity to Radio-Frequency Electromagnetic Radiation

Christopher J. Gordon[1]

I am grateful to the editorial staff of the Journal of Microwave Power for allowing me to respond to the critique of Adair et al. [1983]. The principal objectives of the paper under criticism [Gordon, 1982a] were (i) to interrelate the effects of ambient temperature (T_a) and intensity of exposure to radiofrequency electromagnetic radiation (RFR) on the activity of a single thermoregulatory effector, evaporative heat loss (EHL); and (ii) to ascertain if there is a relation between an animal's body mass and its sensitivity to thermalizing levels of RFR.

In the following response, I shall first explain the rationale and significance of interrelating the effects of T_a versus RFR; second, I shall attempt to demonstrate theoretically why there should be an inverse relation between body mass and sensitivity of the organism to RFR; and third, I shall report additional data that further demonstrate the principle of an inverse relation between body mass and SAR thresholds of physiological activity.

Ambient Temperature vs. SAR

Evaporative heat loss (EHL) was measured in mice exposed to 2450-MHz RFR at a T_a of 20°C because this is within the range of T_a's at which many studies on biological effects of RFR are performed. It is well known that the sensitivity of most thermoregulatory effectors is dependent on T_a. However, my study was designed to assess the effects of RFR at room temperature (20°C), not at T_a's far above or below this level. Any generalization of the EHL data collected at 20°C to other T_a's was never intended or stated. Indeed, I stated in the second-to-last sentence of my paper that variables such as ambient temperature will affect EHL [Gordon, 1982a].

In general, rodents are not well adapted to dissipation of excess thermal energy by evaporation [Hart, 1971; Gordon, 1982b]. However, mice and other rodents will increase the rate of evaporative heat loss (EHL) when thermal loading by endogenous (e.g., exercise) or exogenous sources (e.g., high T_a) is excessive. In my study, exposure of mice to 2450-MHz RFR at SARs of 0 to 29 W/kg for 90 minutes had no measurable effect on EHL. A similar lack of EHL was found in non-exposed mice maintained at T_a's of 20 to 30°C. Above an SAR of 29 W/kg* or above a T_a of 30°C, EHL underwent a significant elevation. These data indicate that, in the mouse, exposure at SARs of 0 to 29 W/kg or to an environment at a T_a of 20°C is thermally equivalent to exposure at T_a's of 20 to 30°C, and raising SAR above 29 W/kg is akin to increasing T_a above 30°C. I believe this to be a significant observation because the study provides one of the first estimates of the amount of RFR-induced heat stress on a thermoregulatory effector relative to the effects of increasing T_a.

A minor point in the paper [Gordon, 1982a] is that, of the additional energy absorbed from RFR at SARs above threshold, 65% of the heat load was dissipated through EHL.† Adair et al. [1983] stated that this value is less when one considers the metabolic production of energy. In my study metabolic compensation was not included in the analysis because the purpose of the paper was to relate the effects of SAR on EHL, not the combined effects of SAR and metabolic rate.

Body Mass vs. Threshold SAR: Theoretical Considerations

An obvious question arises from collection of the EHL data: "Is the threshold SAR necessary to produce EHL in the mouse at a T_a of 20°C the same as that of a larger mammal such as a human being?"

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*The regression line intersected at 25 W/kg but actual data points indicate EHL stability to 29 W/kg.

†The slope of the regression line in Fig. 3 is 0.65 not 0.68. Redrawing of the figure by artist was slightly inaccurate in final print. This has been corrected in an erratum.

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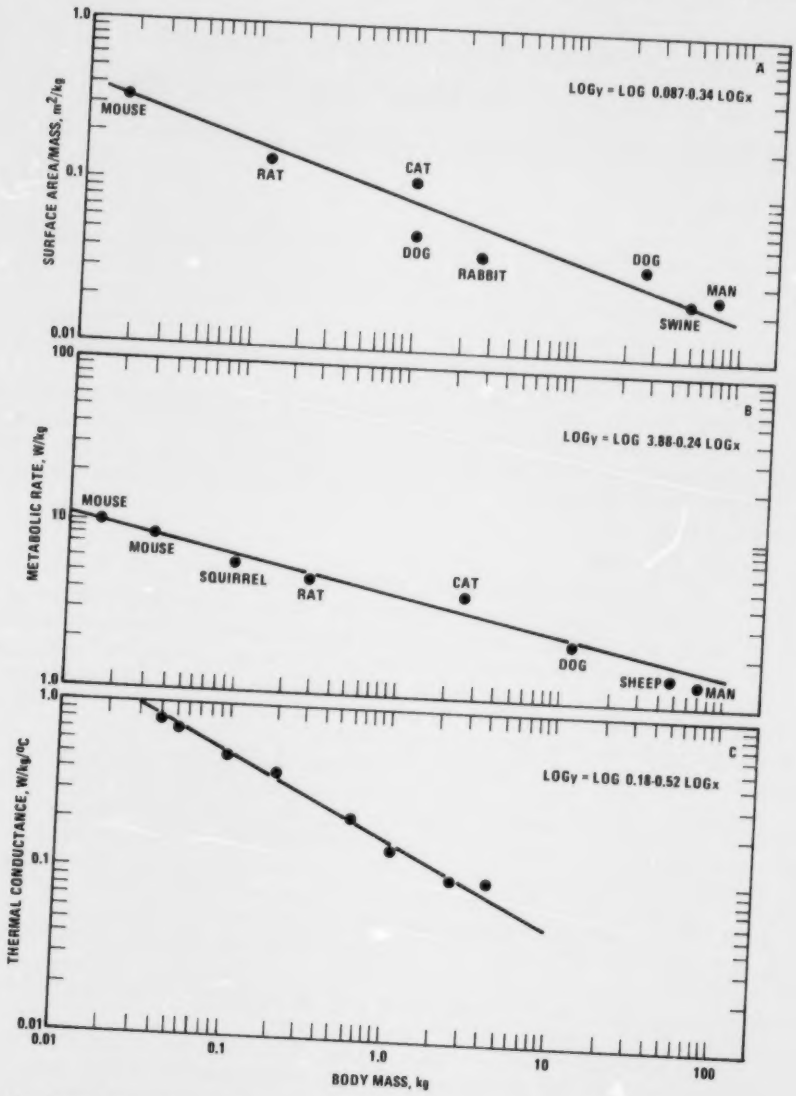

Figure 1 Double logarithmic plots of surface-area to body-mass relation (A); resting metabolic rate at thermoneutrality (B); and minimal thermal conductance vs. body mass (C). Note relative steepness of thermal-conductance regression line compared with surface area and metabolic rate. Data of graph A are modified from Altman and Dittmer, 1962: data of graph B are modified from Schmidt-Nielson, 1979, and data of graph C are from Aschoff, 1981. For sake of clarity, not all data of Aschoff (1981) have been illustrated in graph C.

That is, do small and large mammals have the same threshold SAR for increasing EHL? Analysis of the data in the literature showed that an affirmative assumption of this was incorrect; the data indicate that there is an inverse relation between body mass and the threshold SAR.

An animal's mass or volume is proportional to the cube of its average radial dimension whereas its total surface area (i.e., that which is exposed to the environment) is proportional to the square of the same radial dimension (for discussion, see Schmidt-Nielson [1975]). Thus, the surface area of an

animal increases by the 0.67 power with increasing body mass. The surface-area to body-mass ratio of animals decreases with increasing body mass (Fig. 1A).

A well known fundamental of comparative physiology is that the surface-area to volume ratio of an animal is a significant factor affecting the sensitivity and capacity of the thermoregulatory system. The larger the ratio the greater the amount of passive heat loss. Because of the relation of surface area to volume, small mammals must, to maintain a constant body temperature, generate more metabolic energy per unit of body mass compared with larger species (Fig. 1B). The following examples demonstrate how the surface-area to volume ratio of mammals should greatly affect their sensitivity to RFR.

Example 1. Consider the rate of heat loss per unit body mass of a 0.025-kg mouse and a 70-kg human at a thermoneutral T_a of 28°C. At this T_a both species will maintain a constant core temperature of approximately 37°C. However, the rate of heat loss by the mouse (~9.4 W/kg) is 6.7 times that of the human (~1.4 W/kg). It is important to note that the units of the rate of metabolic energy production and the SAR from RFR are identical (W/kg). If both animals were exposed at a given SAR at properly scaled frequencies of RFR (i.e., such that internal heating patterns were similar; e.g., Durney et al. [1978]), the relative rate of absorbing thermalizing energy would be vastly greater in the human being as compared with the mouse. For example, exposing both species at an SAR of 0.4 W/kg would necessitate an increase of energy dissipation by the human being of 22% and by the mouse of only 4% (assuming both species continued to maintain a core temperature of 37°C).

That energy loading at a given SAR will have a greater effect on the total energy loss in larger mammals is important in relating the threshold SAR to activation of EHL. Taylor [1977], in a comparative analysis of the physiology of exercise, demonstrated an inverse relation between maximal rate of non-evaporative heat loss versus body mass. Taylor calculated that a 0.02-kg animal could dissipate 9 times its metabolic heat production by nonevaporative heat loss, while this would be reduced to just 4.5 times in a 100-kg animal (assuming a 20°C gradient between body and ambient temperature). Of course, one cannot always equate physically or physiologically the thermal loading from exercise and RFR. However, Taylor's analysis does indicate that small mammals can passively dissipate a much greater amount of energy than can larger mammals before EHL is activated. In my study, for example, the mouse absorbed energy at a rate of 29 W/kg before increasing EHL (or an SAR approximately 2.0-times the metabolic rate). If an SAR of just two times the level of metabolic heat production is necessary to increase EHL in the mouse, then application of Taylor's comparative analysis would lead one to expect a much lower threshold SAR to increase active EHL in a 70-kg human being at the same T_a.

Example 2. Because of the surface-area to volume relation, small mammals not only have higher metabolic rates; they also have a greater thermal conductance compared with that of larger mammals (Fig. 1C). In general, the thermal conductance of a 10,000-g mammal is 10 times lower than that of a 100-g species [Aschoff, 1981; Scholander et al., 1950]. In mammals maintained at ambient temperatures below their thermoneutral zone, thermal conductance is maintained at its lowest possible level and can be calculated as:

$$C = M/(T_b - T_a) \tag{1}$$

where,

C = mass specific conductance, $W(kg \cdot °C)^{-1}$
M = metabolic rate, $W \cdot kg^{-1}$
T_a = ambient temperature, °C
T_b = body temperature, °C

by solving for T_b in equation 1,

$$T_b = (M \cdot C^{-1}) + T_a \tag{2}$$

One immediately sees that, if T_a is constant, an increase in metabolic rate will have a greater effect on body temperature in mammals with a lower thermal conductance. Since metabolic rate and SAR are additive it is apparent that a given SAR would be expected to have a greater effect on body temperature in larger mammals. The term C is not fixed and will increase with thermal stress. Increasing C involves physiologic change (i.e., peripheral vasodilation), which is pertinent to this

discussion since we are concerned with threshold SARs that affect a physiological system, not the animal's capacity to dissipate the RFR-induced thermal load. It is important to note that, because conductance is variable, equation (2) cannot be used to predict accurately the body temperature of mammals as a function of increasing SAR (or of increasing metabolic rate). Its use here is intended only to show the general relation between body mass, conductance, and the relative thermal effects of RFR.

The foregoing analysis of body mass versus thermoregulatory sensitivity led to the development of the body-mass versus threshold-SAR regression in Figure 5 of Gordon [1982a]. The procedure was simply a search of the literature for data that bear on a thermoregulatory physiological response in mammals exposed to RFR. One prerequisite of the search was that the SAR value had to be specified or could be calculated accurately. The procedure was to plot the logarithm of body mass versus the logarithm of the threshold SAR for affecting a particular physiological response. Since the ratio of surface-area to volume, the metabolic rate, and the thermal conductance are linearly related to body mass on a log-log plot (Fig. 1), I surmised that threshold SAR versus body mass would also be linearly related by a double logarithmic relationship.

The threshold SAR in some of the studies was provided by the original authors. However, in other cases I had to calculate the threshold based on the data provided. The SAR thresholds shown in Fig. 5 [Gordon, 1982a] are the lowest at which a physiological response to RFR was observed.

Because of the scarcity of data it was necessary to construct the regression line of Fig. 5 on the basis of several physiological endpoints, including evaporative heat loss, metabolism, skin temperature, behavior, and corticosterone production. Adair et al. [1983] questioned the use of data based on the corticosterone research in Dr. Michaelson's laboratory. I used these data because it is well known that a rise in serum corticosterone in the heated mammal is a good index of thermal stress [see, e.g., Follenius et al., 1982]. In addition, the dosimetry in those studies was accurately measured. Regarding related criticisms of Adair et al. [1983], the behavioral thermoregulatory study of Stern et al. [1979] was not included because the experiments were performed on shaved rats maintained in a very cold environment (5°C). Hence, compared with the other studies of Fig. 5, the animals in the study by Stern et al. were in a considerably different ambient environment. The data on metabolism and evaporative heat loss from studies performed in Dr. Adair's laboratory were not included because they were published after my paper went to press.

As stated in my publication, variables such as species, ambient temperature, relative humidity, and carrier frequency, to name a few, will affect the threshold SAR for activating a thermoregulatory response. However, in spite of these variables it is still apparent that small mammals, in general, have a higher threshold SAR than that of larger species [see Fig. 5, Gordon, 1982a]. For example, depending on the T_a and on physiological parameters, studies of the mouse indicate relatively high threshold SARs, ranging from 12 to 45 W/kg. However, studies with rats indicate SAR thresholds of 2 to 7 W/kg, while in the squirrel monkey threshold SARs are approximately 1 to 3 W/kg (see Fig. 2). Of course, these thresholds all will depend on T_a as well as on other factors. But the main point I wish to emphasize is that an SAR of 1.0 W/kg probably will have an effect on a 1-kg squirrel monkey, probably will have little effect on the 0.3 kg rat, and most likely will have no effect on a 0.03-kg mouse. Contrarily, for exposures at resonant RFR frequencies, an SAR threshold of ~20 W/kg for a mouse would invoke a vigorous physiological thermoregulatory response in a rat, and would cause extreme thermal stress and, perhaps, hyperthermic death in the squirrel monkey.

The foregoing discussion is the focal point of Fig. 5 of Gordon [1982a]. The regression line of Fig. 5 simply relates, in a general way, the correlation between body mass and the threshold SAR for EHL. Let me reiterate that, at the time of publication, this concept was primarily descriptive because there had not been enough data collected on a single physiological endpoint in species of differing mass to rigorously define the mathematical relationship. Therefore, I do not want to quibble over the exact values of the intercept and slope of the Fig. 5 regression line as it pertains to predicting the EHL response of human beings to RFR, but instead wish to demonstrate that there most certainly is a strong, inverse dependence of the threshold SAR on body mass.

Scaling the threshold SAR for activating EHL is difficult because of the physiological differences among species. For example, the major avenue of EHL in human beings is the evaporation of sweat on the skin whereas mice and other rodents cannot sweat. The regression line of Fig. 5 predicts an SAR threshold of 0.25 W/kg for active EHL in a human being. However, since ambient temperature in the studies of Fig. 5 varied from 20 to 35°C, the 0.25 W/kg threshold represents a first approximation

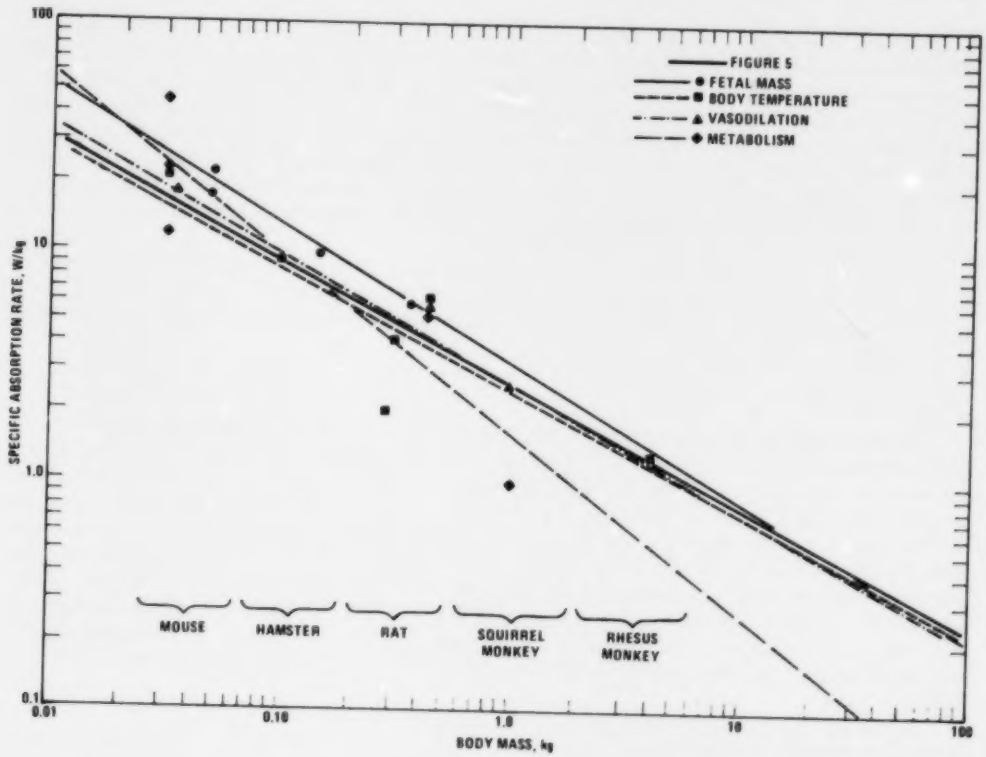

Figure 2 Correlation between threshold of specific absorption rate (SAR) and body mass: for reduction in fetal mass of maternally exposed mice, hamsters, and rats to 2450-MHz RFR [Berman et al., 1978; Berman et al., 1982a; Berman and Carter, 1982b; Berman et al., 1981]; for 0.5 to 1.0°C hyperthermia in mice and hamsters [Berman et al., 1978; Berman et al., 1982] and in rats exposed to 2450-MHz RFR [Phillips et al., 1975; Lotz and Michaelson, 1978; Lu et al., 1981] and in rhesus monkeys exposed to 2.5-MHz RFR [Lotz, 1982]; for peripheral vasodilation in the tail of mice [Gordon, unpublished], of rats [Phillips et al., 1975] and of squirrel monkey exposed to 2450-MHz RFR [Adair and Adams, 1980]; and for reduction in metabolic rate in mice [Ho and Edwards, 1977, 1979], rats [Phillips et al., 1975] and squirrel monkeys exposed to 2450-MHz RFR [Adair and Adams, 1982]. Also plotted is the regression line originally published in Figure 5 of Gordon 1982a. Ambient temperature in all studies (excluding that used to generate data in Figure 5) ranged from 20 to 24°C. Linear regression equation for fetal mass, $\log = \log 3.46 - 0.53 \log X$; body temperature, $\log Y = \log 2.45 - 0.52 \log X$; vasodilation, $\log Y = \log 2.69 - 0.56 \log X$; metabolism, $\log Y = \log 1.58 - 0.77 \log X$.

that will be influenced by ambient temperature. It is of interest to note that the threshold SAR for activating sweating (~0.45 W/kg at 200 MHz and ~0.2 W/kg at 80 MHz) in a thermal model of a human being at an ambient temperature of 30°C developed by Spiegel (cited in Gordon, 1982a) falls very close to the predicted value in Fig. 5 (0.25 W/kg). Lowering the ambient temperature to 20°C will raise the threshold SAR.

Body Mass vs. Threshold SAR: Additional Experimental Evidence

As mentioned earlier, a difficulty in accepting the principle of Figure 5 [Gordon 1982a] is that the experimental data on various physiological endpoints were collected under a variety of ambient conditions. Further demonstration of an inverse relation between body mass and threshold SAR requires the measurement of a single physiological parameter in species of well distributed masses under similar environmental conditions.

Figure 2 is a log-log plot of threshold SAR versus body mass of several endpoints including inhibition of metabolism, vasodilation, hyperthermia (levels of 0.5 to 1.0°C), and hyperthermically associated reduction in fetal mass. Ambient temperature in all the studies of Fig. 2 ranged from 20 to 24°C. Thus, the regression lines in Fig. 2 are more specific than that originally published since each line represents a single physiologic endpoint measured within a relatively narrow range of T_a's ($\leq$4°C in the present analysis compared with a span of 15°C in the original study). The inverse relation between body mass and threshold SAR is obvious under all endpoints measured. It is interesting to note that, for a 70-kg human being, the regression lines extrapolate to a threshold SAR of 0.06 to 0.3 W/kg, depending on the physiological endpoint.

It should be noted, with regard to the comments of Adair et al. [1983], that additional metabolic data from Ho and Edwards [1979] have been added to the metabolism regression line of Fig. 2. In my opinion these data (12 and 45 W/kg) do not "fall well off the regression line" as claimed by Adair et al. [1983].

CONCLUSION

Regarding the last paragraph of the critique by Adair et al. [1983], I agree that we have a very good understanding and can quantify the thermoregulatory responses of human beings, but only in situations of exposure to conventional sources of thermal energy (e.g., high T_a, infrared irradiation, and exercise). There are few data, and it is doubtful that there ever will be data collected on the thermoregulatory responses of human beings under unrestricted exposure of the body to RFR. Therefore, I must contend that in the study of physiological effects of RFR, we understand the thermoregulatory responses of laboratory animals far better than we understand those of human beings. Furthermore, if we are to relate the physiological effects of RFR on laboratory animals to man, and since laboratory animals (i.e., rodents, rabbits, and primates) are 1 to 3.5 orders of magnitude smaller than human beings, we must understand thoroughly the relation between body mass and sensitivity to RFR.

I disagree with the statement of Adair et al. [1983] that ". . . no relationship such as that depicted in Figure 5 [Gordon, 1982a] can possibly exist." The data of Fig. 5 and in Fig. 2 here show that there is an inverse relation between threshold SAR and body mass for laboratory mammals ranging in mass from 0.03 to 4.0 kg. Of course, because of physiological differences between human beings and laboratory mammals, one must be cautious in extrapolating the data of infrahuman species to man. However, in spite of the physiological differences, it is apparent from the foregoing discussion that body mass is a critical parameter in governing the sensitivity of mammals to RFR.

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